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=> s 14 and (cleavage)

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L5 ANSWER 1 OF 6 BIOSIS COPYRIGHT (c) 2006 The Thomson Corporation on STN

TI Regulation of fibrinolysis by non-esterified fatty acids.

AB The ability of oleic acid to modulate fibrinolysis was me

The ability of oleic acid to modulate fibrinolysis was measured by following the urokinase-mediated and plasminogen-dependent cleavage of 125I-labelled fibrin clots. Oleic acid levels within the physiological range exerted a concentration-dependent inhibition of urokinase-mediated fibrinolytic activity. SDS/PAGE revealed that oleic acid enhances urokinase activity but simultaneously increases the autolytic cleavage of the newly formed low-molecular-mass subunit of plasmin. Oleic acid-induced cleavage of this subunit containing the catalytic site of plasmin was suppressed by the plasmin substrate H-D-valyl-L-leucyl-L-lysine-p-nitroanilide (S-2251) and was prevented by alpha-2-antiplasmin. A concentration-dependent inhibition of the activity of purified plasmin on 125I-labelled fibrin clot was also observed; 93% and 50% inhibition was noted with 150 mu-M and 32 mu-M oleic acid respectively. Oleic acid at 200 mu-M also effectively displaced plasmin prebound to a polylysine-Sepharose column. Examination of the fatty acid specificity showed that a minimal chain length of 16 carbon atoms and the presence of at least one double bond, preferably in a cis configuration, were required for inhibition of the fibrinolytic activity of plasmin. Oleic acid at a concentration that produced only a minimal inhibition of plasmin activity induced a marked inhibition by palmitic acid, while palmitic acid alone is ineffective. The findings suggest that oleic acid stimulates plasminogen activation and modulates the fibrinolytic and autolytic activities of plasmin.

ACCESSION NUMBER:

1994:310531 BIOSIS

DOCUMENT NUMBER:

PREV199497323531

TITLE:

Regulation of fibrinolysis by non-esterified fatty acids.

AUTHOR (S): Higazi, Abd Al-Roof [Reprint author]; Aziza, Rifat; Samara,

Abd Al-Ruhman; Mayer, Michael

CORPORATE SOURCE: Dep. Clin. Biochem., Hadassah Med. Cent., P.O. Box 12000,

Jerusalem, IL 91120, Israel

Biochemical Journal, (1994) Vol. 300, No. 1, pp. 251-255. SOURCE:

ISSN: 0264-6021.

DOCUMENT TYPE: LANGUAGE:

Article English

ENTRY DATE:

Entered STN: 26 Jul 1994

Last Updated on STN: 27 Jul 1994

L5 ANSWER 2 OF 6 BIOSIS COPYRIGHT (c) 2006 The Thomson Corporation on STN

RELEASE OF B-BETA PEPTIDES FROM FIBRINOGEN OR FIBRIN IN THE PRESENCE OF ΤI

ALPHA ANTIPLASMIN.

AB When Glu-plasminogen (plg) was activated by urokinase (UK) in the presence of fibrinogen or fibrin, Bβ peptides (Bβ 1-42) were released faster from fibrinogen than from fibrin(Bβ 15-42). These results were contrary to faster release of $B\beta$ 15-42 from fibrin in the UK-activated clotted plasma in comparison to the release of Bß 1-42 from UK-activated plasma. The addition of plasma or lysine-Sepharose pass through fraction to the above system resulted in faster release of $B\beta$ peptides from fibrin than fibrinogen. The addition of .alpha. 2 antiplasmin (α 2AP) to the mixture of Glu-plq, UK and fibrinogen or fibrin resulted in faster release of $B\beta$ peptides from fibrin than from fibrinogen. These results indicate that fibrin protected plasmin from inactivation by $\alpha 2AP$, leading to cleavage of Arg(42)-Ala(43) bond in β -chain of fibrin which seems to be less susceptible to plasmin than the same bond in fibrinogen.

ACCESSION NUMBER: 1986:255632 BIOSIS

PREV198682010381; BA82:10381 DOCUMENT NUMBER:

TITLE: RELEASE OF B-BETA PEPTIDES FROM FIBRINOGEN OR FIBRIN IN THE

PRESENCE OF ALPHA ANTIPLASMIN.

AUTHOR(S): TAKADA A [Reprint author]; MAKINO Y; TAKADA Y

CORPORATE SOURCE: DEP PHYSIOLOGY, HAMAMATSU UNIV, SCH MED, HAMAMATSU-SHI.

SHIZUOKA-KEN, JPN

Thrombosis Research, (1986) Vol. 42, No. 1, pp. 1-10. SOURCE:

CODEN: THBRAA. ISSN: 0049-3848.

DOCUMENT TYPE:

FILE SEGMENT: BA

LANGUAGE: **ENGLISH**

ENTRY DATE: Entered STN: 21 Jun 1986

Article

Last Updated on STN: 21 Jun 1986

L5 ANSWER 3 OF 6 BIOSIS COPYRIGHT (c) 2006 The Thomson Corporation on STN TI EFFECTS OF HUMAN GRANULOCYTE ELASTASE ON FIBRINOLYSIS.

AB The specific limited proteolysis of human plasminogen by human granulocyte elastase results in the cleavage of 3 peptides from the H chain of plasminogen. These 3 peptides bind to lysine- and fibrin-Sepharose. The remainder of the molecule possesses a MW of 38,000 and represents a fully activatable plasminogen lacking the lysine binding site(s) in the H chain. Upon treatment of this low MW form of plasminogen with urokinase, esterolytic, fibrinolytic and amidolytic activities appear. The low MW form of plasmin consists of 2 S-S-linked polypeptide chains with MW .apprx. 25,000 and 13,000. The kinetic parameters of hydrolysis of synthetic substrates by the low MW form of plasmin and native plasmin, respectively, are similar. The inhibitions of the 2 enzymes by several inhibitors of the hydrolysis of Blue Dextran-fixed fibrin are identical. The initial rate of activation with urokinase is .apprx. 2 times faster for the low MW form of plasminogen than for native plasminogen, and the activation of native plasminogen by urokinase in inhibited by plasma . alpha.2-antiplasmin, whereas the activation of the low MW form of plasminogen is not. Urokinase-induced clot lysis is inhibited efficiently by .alpha.2-antiplasmin, but the

inhibitory effect of .alpha.-antiplasmin is removed by H chain fragments containing lysine binding site(s), which are obtainable from plasminogen by limited elastase digestion. Urokinase-induced fibrinolytic activity of plasma is promoted by human granulocyte elastase in an apparently time-dependent manner. Apparently, lysine binding site(s) in the H chain of plasmin(ogen) are of great importance in relation to the rate of its reaction with .alpha.2

-antiplasmin.

ACCESSION NUMBER: 1982:147824 BIOSIS

DOCUMENT NUMBER: PREV198273007808; BA73:7808

EFFECTS OF HUMAN GRANULOCYTE ELASTASE ON FIBRINOLYSIS. TITLE:

NAGAMATSU A [Reprint author]; SOEDA S AUTHOR(S):

CORPORATE SOURCE: FACULTY OF PHARMACEUTICAL SCIENCES, FUKUOKA UNIV, NANAKUMA,

NISHI-KU, FUKUOKA, 814-01, JAPAN

SOURCE: Chemical and Pharmaceutical Bulletin (Tokyo), (1981) Vol.

29, No. 4, pp. 1121-1129.

CODEN: CPBTAL. ISSN: 0009-2363.

DOCUMENT TYPE:

FILE SEGMENT: BA

LANGUAGE: ENGLISH

ANSWER 4 OF 6 USPATFULL on STN 1.5

ΤI Compositions and methods for treating thrombotic disorders

The invention provides compositions containing a chimeric microplasmin AB

polypeptide and methods for fibrinolytic therapy using the chimeric

polypeptide.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

ACCESSION NUMBER: 2004:299248 USPATFULL

TITLE: Compositions and methods for treating thrombotic

disorders

INVENTOR(S): Reed, Guy L., Boston, MA, UNITED STATES

NUMBER KIND DATE -----PATENT INFORMATION: US 2004235115 A1 20041125 APPLICATION INFO.: US 2003-715981 A1 20031118 (10)

NUMBER DATE

PRIORITY INFORMATION: US 2002-427152P 20021118 (60) DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION

LEGAL REPRESENTATIVE: MINTZ, LEVIN, COHN, FERRIS, GLOVSKY, AND POPEO, P.C.,

ONE FINANCIAL CENTER, BOSTON, MA, 02111

NUMBER OF CLAIMS: 2.0 EXEMPLARY CLAIM:

NUMBER OF DRAWINGS: 5 Drawing Page(s)

LINE COUNT: 1041

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L5ANSWER 5 OF 6 USPATFULL on STN

TI Yeast expression vector and a method of making a recombinant protein by expression in a yeast cell

Vectors for the expression in yeast of mammalian plasminogen derivatives AΒ such as microplasminogen and miniplasminogen are presented. Methods for expression of these proteins in a methylotrophic yeast expression system are disclosed as well as the activation and stabilisation of the recombinant proteins. The proteins of this invention are used In the treatment of focal cerebral ischemic infarction and other thrombotic diseases.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

ACCESSION NUMBER:

2004:94224 USPATFULL

TITLE:

Yeast expression vector and a method of making a recombinant protein by expression in a yeast cell

INVENTOR(S):

Collen, Desire Jose, London, UNITED KINGDOM

Nagai, Nubuo, Leuven, BELGIUM Laroche, Yves, Brussel, BELGIUM

		NUMBER	KIND	DATE	
PATENT INFORMATION:	US	2004071676	A1	20040415	
APPLICATION INFO.:	US	2003-450976	A1	20031208	(10)
	WO	2001-BE217	•	20011220	

DOCUMENT TYPE: FILE SEGMENT:

Utility APPLICATION

LEGAL REPRESENTATIVE:

CLARK & ELBING LLP, 101 FEDERAL STREET, BOSTON, MA,

02110

NUMBER OF CLAIMS: EXEMPLARY CLAIM: 41

NUMBER OF DRAWINGS:

5 Drawing Page(s)

LINE COUNT:

1656

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L5 ANSWER 6 OF 6 USPATFULL on STN

TI Methods of inhibiting the activation of Factor XIII

AB Compositions and methods for inhibiting the activation and/or active state of a precursor protein are provided. Compositions provided can bind to a cleavage site of a precursor protein. Also provided are compounds useful for generating inhibitor compositions. Application of the invention to the treatment of myocardial infarction and other thrombotic conditions is specifically provided. Further provided are antibodies specific for active Factor XIII.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

ACCESSION NUMBER:

97:31410 USPATFULL

TITLE:

Methods of inhibiting the activation of Factor XIII

INVENTOR (S):

Reed, Guy L., Winchester, MA, United States Matsueda, Gary R., Princeton, NJ, United States

Haber, Edgar, Salisbury, NH, United States

PATENT ASSIGNEE(S):

Bristol-Myers Squibb Company, Princeton, NJ, United

States (U.S. corporation)

General Hospital Corporation, The, Boston, MA, United

States (U.S. corporation)

	NUMBER	KIND DATE	
PATENT INFORMATION:	US 5620688	19970415	
	WO 9215609	19920917	
APPLICATION INFO.:	US 1994-117052	19940504	(8)
	WO 1992-US1926	19920311	
		19940504	PCT 371 date
		19940504	PCT 102(e) date

19940504 PCT 102(e) date
Continuation-in-part of Ser. No. US 1991-667296, filed

RELATED APPLN. INFO.: Continuation-in-part of Ser. No. on 11 Mar 1991, now abandoned

DOCUMENT TYPE: Utility FILE SEGMENT: Granted

PRIMARY EXAMINER: Chan, Christina Y. ASSISTANT EXAMINER: Gambel, Phillip

LEGAL REPRESENTATIVE: Sterne, Kessler, Goldstein & Fox P.L.L.C.

NUMBER OF CLAIMS:

EXEMPLARY CLAIM: NUMBER OF DRAWINGS:

1 Drawing Figure(s); 1 Drawing Page(s)

LINE COUNT:

1599

CAS INDEXING IS AVAILABLE FOR THIS PATENT.